Asthma and Cardiac Dyspnea—A Differential Diagnosis

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SUMMARY

There appear to be no infallible guides by which to differentiate between cardiac insufficiency and asthma as a cause of dyspnea, wheezing and coughing in elderly patients. Many of the symptoms of one condition are also symptoms of the other. Even the results of therapeutic trial cannot be relied upon to establish diagnosis, for drugs effective in treatment of heart disease may also help relieve asthma, and vice versa.

Although there is no single factor that can be considered pathognomonic, there are certain symptoms and results of tests which are more strongly indicative of one condition than of the other. Careful evaluation of all factors, while it may not serve to establish unequivocal diagnosis, will provide a basis for judicious treatment of the patient.

OFTEN in the case of elderly patients with dyspnea, coughing and wheezing, it is difficult to determine whether the symptoms are ascribable to bronchial disease or to cardiac insufficiency. In considering the problem, it is well first to establish what is meant by the terms bronchial asthma and "cardiac asthma."

Swineford and Magruger[8] adopted the purist's point of view, that asthma develops only in persons with allergic sensitivity and heart failure. Miller,[3] on the other hand, distinguished between bronchial asthma due to cardiac disturbance and bronchial asthma due to other causes. The purposes of distinction would appear to be best served by discarding the term "bronchial" and using the term "asthma," in accord with standard nomenclature, as meaning the disorder in the bronchioles usually associated with the phenomena of allergy. And the cardiac disorder which so often causes symptoms much like those of asthma is paroxysmal left ventricular failure, most frequently due to hypertensive or arteriosclerotic cardiovascular disease, with varying degree of impaired coronary blood flow.

The typical attack of dyspnea of cardiac origin occurs at one or two o'clock in the morning, awakening the patient after two to four hours of sound sleep. Shortness of breath and wheezing, and a sense of a weight on the chest, cause the patient to sit up in bed. Often the attack is relieved in a few minutes by the coughing up of tenacious mucus, and the patient then returns to sleep for the remainder of the night. More severe attacks of dyspnea and wheezing may last minutes to hours; some may, of course, progress to pronounced pulmonary edema and death. The attacks may be infrequent, or they may occur nightly. In the latter case, the patient dreads going to bed and finds that he gets more rest spending the night in a chair.

Patients with asthma, in which the infectious factor predominates, often have quite similar symptoms. They, too, are awakened usually at three or four o'clock in the morning with dyspnea, wheezing and coughing, and relief usually follows the raising of a small amount of tenacious mucus which has been dislodged by inhalation of the fumes of burning stramonium leaves or by taking one of the sympathomimetic drugs. They, too, are comfortable during the day and dread the nights with the recurrence of paroxysms.

The primary mechanisms by which the symptoms in these two disorders develop are quite dissimilar. In the asthmatic patient, the primary changes are concerned with encroachment upon the lumen of the bronchiole, with changes in the musculature and in the submucosa, with an increase in the number of mucus-secreting glands, with changes in the mucosa itself, and with the accumulation of mucus and cellular elements in the already narrowed lumen. In the patient with respiratory distress of cardiac origin, a more elaborate succession of events has been described,[1, 2] following upon the primary failure of the left ventricle. In it are concerned decreased serum protein, changes in plasma volume, and an increase in blood volume in the pulmonary circuit. After the patient has been recumbent for several hours, the factors leading up to pulmonary edema manifest themselves as the characteristic symptoms. In many instances, after a period of time, the patient also has well-established changes in the bronchioles, especially in the submucosa, and the accumulation of inspirated mucus in the somewhat narrowed lumen causes the perplexing wheeze.

The determination of which mechanism is primarily responsible in the development of dyspnea and wheezing is extremely important with regard to prognosis and treatment. One great cause of difficulty in differential diagnosis is the attempt to attribute symptoms to either one mechanism or the other. Often, in preplexing cases, the patient has both primary bronchial and cardiac disease. Riley[6] aptly stated that when symptoms of both pulmonary and cardiac origin coexist, as frequently happens in the older age group, the clinical evaluation of each with respect to its contribution to the dyspnea and wheezing may be extremely difficult, if not impossible.

Short clinical reports on the cases of two elderly patients with dyspnea, coughing and wheezing will illustrate the point.

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CASE REPORTS

CASE 1: A 70-year-old mortician was first observed in June 1947 with complaint of coughing, wheezing and dyspnea, chiefly at night, and particularly persistent in the previous three months. The patient had had perennial asthma at age 19, while living in Ohio. The attacks diminished in frequency and severity after he moved to the Pacific Northwest.

Upon examination it was noted that the lung fields were clear and there was no peripheral edema. The blood pressure was at all times within normal limits. In fluoroscopic examination, cardiac enlargement of aortic configuration was observed, with flattened diaphragms and evidence of some pulmonary fibrosis and emphysema. Seventeen per cent of the leukocytes in the sputum were eosinophils. In preliminary testing with common inhalants there was significant reaction to feathers and to house dust. An electrocardiographic tracing was definitely abnormal, with evidence of bundle branch block the principal feature (Figure 1). Clinical symptoms, however, suggested asthma due probably to both inhalant and infectious factors. Symptomatic treatment with iodides and aminophylline was given and further allergic sensitivity studies were begun. However, the respiratory distress became more pronounced and more continuous. The pulse rate at rest remained at 96, and the clinical symptoms became more apparently those of failing left ventricle. Appropriate therapy was instituted, including bed rest, low sodium intake and digitalization. Within three days the patient became asymptomatic. There was moderate diuresis with loss of eight pounds or more in body weight. As the patient then was able to resume greater activity, allergic sensitivity studies were completed and treatment with autogenous vaccine and the ubiquitous inhalants was started. A maintenance dose of 0.1 gm. of digitalis leaf daily was given, and after several months, the previous conduction defect was absent from electrocardiographic tracings (Figure 1). In the ensuing three and one-half years the patient worked daily at his profession. In that period of time he was observed twice in what appeared to be typical asthmatic attacks following a respiratory tract infection. Each attack responded within 24 hours to iodide given orally and one intravenous injection of aminophylline.

CASE 2: An 84-year-old widow was referred, with a diagnosis of asthma, by an internist. As the patient was obviously senile, there was ground for suspicion that advanced degenerative changes in the cardiovascular system were responsible for the respiratory distress. The patient had had attacks of coughing, wheezing and dyspnea over a period of 24 years. The attacks occurred at irregular intervals, without apparent relation to season or common allergens, and sometimes were so severe as to cause transient loss of consciousness. The patient recalled having had seasonal hay fever many years ago, while living in the Puget Sound area, and symptoms of chronic perennial rhinitis with recurring nasal polyps. Bacterial vaccine therapy and change of climate had given no relief. Epinephrine, by injection, was no longer effective; and inhalation of 1 per cent epinephrine solution, once effective, was becoming less so.

On physical examination, moderate tachycardia and evidence of moderate emphysema were noted. The blood pressure was 130 mm. of mercury systolic and 60 mm. diastolic. No significant abnormalities were noted in auscultation of the heart. Five per cent of the peripheral blood cells were eosinophils but there was no eosinophilia in the sputum. In an electrocardiographic tracing (Figure 1) there was a well-established bundle branch block.

As the dyspnea and wheezing appeared, from this evidence, to be caused by cardiac insufficiency, a low sodium diet was prescribed, and digitoxin (on a dosage schedule for slow digitalization) and aminophylline were given. The patient

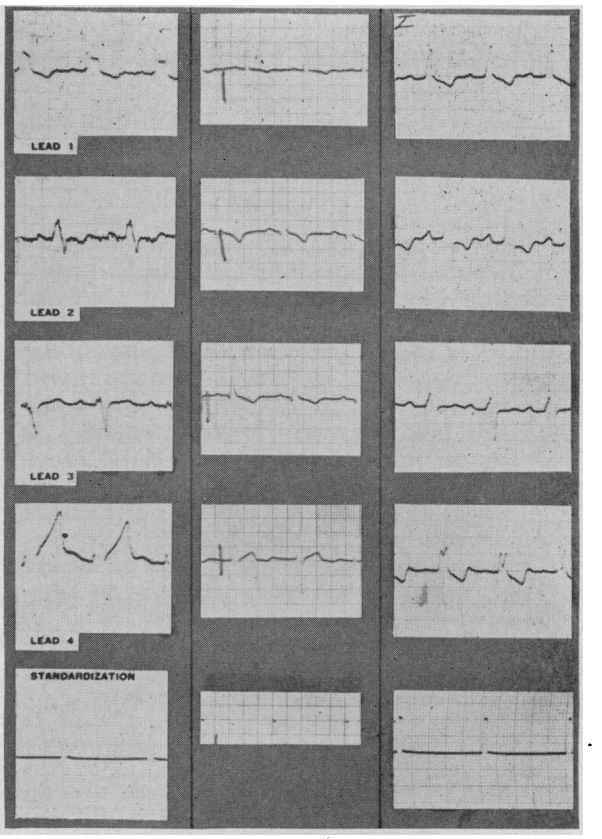

Figure 1.—*Left:* Electrocardiogram (Case 1) taken July 2, 1947. *Center:* Electrocardiogram of same patient, April 22, 1948. *Right:* Electrocardiogram (Case 2) taken May 13, 1948.

was admonished to discontinue the use of sympathomimetic drugs, especially by inhalation.

Despite conscientious cooperation, there was no improvement, and attacks continued to occur nightly. Reevaluation of the probable cause of wheezing and dyspnea was indicated. Since the digitoxin caused no reduction in pulse rate, or diuresis, or symptomatic improvement, the drug was discontinued. A regimen with potassium iodide, small doses of ephedrine and an occasional subcutaneous injection of epinephrine was started. The patient then improved slowly. When last observed, after a year on this regimen, the patient had had mild respiratory distress only occasionally after respiratory tract infection. She was taking one or two half-tablets of Tedral® a day, and an injection of 0.2 cc. of epinephrine once or twice a month. The chief night distress was moderate nasal obstruction which was controlled by use of an antihistaminic drug.

The original diagnoses of the primary cause of the respiratory distress in these two cases were far from correct. In the first case, the preponderance of evidence seemed to indicate asthma. Yet, at the time of acute recurrent respiratory distress, there was dramatic response to treatment for cardiac disease.

In the second case, in which there was no good evidence of asthma, heart failure seemed most probably the cause of respiratory distress. However, there was no improvement when adequate treatment for cardiac insufficiency was carried out, but

good response when iodides and sympathomimetic drugs were given.

In many instances the final recourse in the differentiation of the cause for paroxysmal respiratory distress in an elderly patient is therapeutic trial. Fortunately for the patient, certain drugs, especially the soluble theophylline preparation and iodides, cause little or no undesirable side effects, and the former may help to release both asthma and cardiac dyspnea. Epinephrine, subcutaneously, may also relieve cardiac dyspnea,[5] although much less dramatically than it relieves asthma. As the alternate effectiveness of these drugs only adds to the confusion, even therapeutic trial cannot be relied upon for differentiation. Although there are no infallible guides, there are certain observations and procedures that will help toward a better evaluation of the cause for dyspnea and wheezing in elderly patients and provide a basis for more judicious treatment.

Indicative of asthmatic origin:

1. A long history of asthma or other allergic diseases.
2. Other manifestation of allergic reaction.
3. Sputum tenacious and containing Charcot-Leyden crystals, eosinophils, and Curschmann spirals.
4. Eosinophilia in blood and secretions (nasal and bronchial).
5. Circulation time normal or even shortened.
6. Response, often dramatic, to iodides and sympathomimetic drugs.

Indicative of cardiac origin:

1. History or evidence of cardiovascular-renal disease.
2. Moist basilar rales in addition to sonorous and sibilant rales.
3. Sputum more fluid or frothy and even blood-tinged, without Charcot-Leyden crystals or eosinophils.
4. Respirations more rapid and the two phases less disproportioned.
5. Circulation time prolonged (whether asthma coexists or not).[4]
6. Good response to treatment for cardiac insufficiency (including bed rest, digitalis, and mercurial diuretics).[7]

DISCUSSION

In the enumeration of points indicative of each of the causes of respiratory distress, it is quite evident that there is no pathognomonic factor by which to establish a single etiologic diagnosis. Prolonged circulation time would seem to be the factor most strongly indicative of cardiac disease and eosinophilia of asthma; yet often in obviously asthmatic patients eosinophilia is not present, and often in the presence of unquestionable paroxysmal left ventricular failure there is no significant lengthening of circulation time. And so it becomes evident that a frequent error is failure to keep in mind that in mild congestive heart disease an intrinsic bronchial disorder may become prominent. On the other hand, it must not be overlooked that a patient with a long history of bronchial asthma is just as liable to cardiovascular disease as is a patient who never had asthma.

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